

**REMARKS**

Claims 1, 3, 5 to 7 are pending in the application. Claims 2, 4, 8 to 11 and 13 have been previously cancelled. Claim 12 has been cancelled by the present amendment.

*Claim Rejection – 35 USC § 102*

Claim 12 has been cancelled, thereby rendering the rejection moot.

*Claim Rejection – 35 USC § 103(a)*

Claims 1, 3 and 5 to 7 have been rejected under 35 USC § 103(a) as being allegedly obvious in view of Freeze et al.

Applicant respectfully disagrees and submits that Freeze never disclosed or suggested to use antibodies directed against the S100A proteins to inhibit the recruitment and/or activation of neutrophils. Applicant submits that Freeze has clearly shown that glycans present on endothelial cells do bind with the several protein targets present on neutrophils (such as S100A8, S100A9, amphoterin and annexin 1). Applicant also submits that Freeze has shown that an antibody directed against the glycans present on the endothelial cell (antibody mAbGB3.1) and an antibody against S100A9 does inhibit the interaction between endothelial cells and neutrophils. But Applicant disagrees with the Examiner and submits that Freeze does not show or suggest that the antibody against S100A9 does inhibit the extravasation of neutrophils, the recruitment of neutrophils or the inflammatory process. Freeze has only shown that antibodies directed against S100A9 disrupt the interaction between endothelial cells and neutrophils but has failed to show or suggest that the same antibodies can be used for inhibiting the extravasation of neutrophils, recruitment of neutrophils or inflammation.

The extravasation process is a complex one that does not solely involve the binding of the neutrophils to endothelial cells. It has been clearly shown in the art of immunology that, even though endothelial cells and neutrophils are known to interact with one through their own cell membrane targets, the inhibition of interaction between the two targets does not necessarily lead to the inhibition of extravasation. As an example, the Applicant herewith submits a scientific publication by Perretti et al. (submitted in the enclosed I.D.S. document), where it is clearly shown that the inhibition of interaction between annexin 1 (also known as lipocortin 1)

and its target on endothelial cells augments extravasation of neutrophils. To this effect, the Examiner is referred to Figure 3, panel *b*, where it is shown that neutralizing antibody mAb 1A augments the extravasation of neutrophils. The Examiner will appreciate that annexin 1 was identified by Freeze as a possible target to the glycans present on endothelial cells. Therefore, the Examiner's prediction that the inhibition of neutrophil cell membrane targets recognized by the glycans taught by Freeze would necessarily lead to the inhibition of neutrophil extravasation is erroneous.

Applicant respectfully submits that the prior art teaches away from the claimed subject matter prior to the filing of the present application. Freeze has identified glycans present on endothelial cells that, when neutralized by mAbGB3.1, inhibit the extravasation of neutrophils. Freeze has also shown that the glycans recognized by the monoclonal antibody can bind to the several target proteins such as S100A8, S100A9, amphoterin and annexin 1. Freeze himself has shown that antibodies directed against S100A8 are not capable of limiting neutrophil extravasation. Perretti et al. (cited *supra*) has also shown that antibodies directed against annexin 1 augment neutrophil extravasation. Therefore, the prior art teaches away from inhibiting the neutrophil cell membrane target to limit extravasation and/or inflammation. In light of the above, the Applicant respectfully submits that because the prior art submitted herewith teaches away from the claimed subject matter, the claims presently on file are non-obvious (*In re Fine*, 387 F. 2d 1071, 5 USPQ2d 1596 (Fed Cir. 1988)).

Applicant further submits that, because the prior art teaches away from the claimed subject matter, the person skilled in the art would not be motivated to try, with a reasonable chance of success, inhibiting the neutrophil protein S100A9 to limit neutrophil extravasation, recruitment and/or activation. In light of the above, and because the immunological art is a very difficult art to predict, Applicant respectfully submits that the claims presently on file are non-obvious (*In re Tomlinson*, 150 USPQ 623 (CCPA 1966)).

Applicant submits that no new matter has been added by the present amendments.

It is submitted, therefore, that the claims are in condition for allowance. Reconsideration of the Examiner's rejections is respectfully requested. Allowance of claims 1, 3 and 5 to 7 at an early date is solicited.

In the event that there are any questions concerning this amendment or the application in general, the Examiner is respectfully urged to telephone the undersigned so that prosecution of this application may be expedited.

No additional fees other than the fees for filing an IDS are believed to be associated with the filing of this amendment. However, should this assumption be an error, the Commissioner is hereby authorized to charge the required fee to Deposit Account No. 19-5113.

Respectfully,

UNIVERSITÉ LAVAL



Marie-Hélène Rochon, Registration No. 57,566  
Agent of the Applicant

December 15, 2006

OGILVY RENAULT, LLP/S.E.N.C.R.L., s.r.l.  
1981 McGill College Ave.  
Suite 1600  
Montréal, Québec  
Canada H3A 2Y3  
Tel. : (514) 847-6095 / Fax : (514) 288-8389